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Emergency Medicine Practice
Evidence-Based Education • Practical Application

CLINICAL CHALLENGES:

- **What are the key features** that distinguish high-risk syncope from low-risk syncope?
- **When should risk-stratification tools** be used?
- **When is hospitalization** appropriate?

Emergency Department Management of Syncope

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Prior to beginning this activity, see "CME Information" on page 22.

Abstract

Syncope is the transient loss of consciousness and postural tone, with spontaneous recovery. It accounts for approximately 1% of all emergency department visits and $5.6 billion in healthcare costs annually. In a very small subset of patients, syncope may be a warning sign for serious outcomes or death, but identifying these patients is challenging, as the emergency clinician must distinguish between life-threatening causes and the more common, benign etiologies. Low-yield and expensive testing is often performed, even for benign presentations. Much research on syncope is observational, and clinical decision rules frequently perform poorly in validation studies. This issue reviews the clinical and diagnostic findings that are useful for safely and efficiently identifying patients presenting to the emergency department with syncope.

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Case Presentations

CASE 1

A 16-year-old girl is referred to the ED for a head CT after passing out in a doctor's office…

- As you review the chart, you note that the girl was in her dermatologist's office, undergoing excision of a mole, when she became lightheaded and diaphoretic and passed out, without striking her head.
- She was unconscious less than 1 minute and awoke with no confusion.
- Because she passed out, the dermatologist told the parents that they should "go immediately to the ER for a CT." You wonder if that's really necessary in this case…

CASE 2

A 40-year-old man presents by EMS after what bystanders describe as a "seizure" while he is sitting in a chair…

- EMS reports that bystanders told them that the man lost consciousness while sitting in a chair, and that he had generalized jerking of his extremities. When EMS arrived a few minutes later, they noted that his mental status was normal, but he was complaining of chest pain and shortness of breath.
- The patient says that he underwent elective cholecystectomy 3 weeks ago, but denies abdominal pain or vomiting, and he says he has no other medical problems.
- His vital signs are: blood pressure, 96/54 mm Hg; heart rate, 122 beats/min; respiratory rate, 24 breaths/min; temperature, 37°C; and oxygen saturation, 90% on 2 L nasal cannula.
- The nurse asks if you want to place the patient on seizure precautions, but you're not sure that's his major underlying problem…

CASE 3

An 80-year-old woman presents to the ED after a fall…

- The patient states she was walking to the bathroom when she found herself on the floor with right knee pain, and no recollection of how she got there. She currently has no other complaints.
- Her vital signs are normal, and her past medical history is significant for diabetes, hypertension, and heart failure.
- The x-rays ordered at triage are normal, and she is able to ambulate, but you wonder if there's something else going on that you should look into before she leaves…

Introduction

Resulting from global cerebral hypoperfusion, syncope is a transient loss of consciousness (TLOC), typically with associated loss of postural tone, followed by complete, spontaneous recovery. Syncope represents approximately 1% of all emergency department (ED) visits in the United States, with around one-third of those patients being admitted to the hospital.[1] Although there has been extensive research on this condition, the proportion of patients admitted has remained stable for the last 15 years, and despite increased resource utilization, the most common discharge diagnosis remains the symptomatic diagnosis, "syncope and collapse."[2] Annual expenditures for syncope admissions are estimated at up to $5.6 billion in the United States.[3] For most patients with syncope, hospitalization has a much smaller impact on mortality than the patient's underlying morbidities.[4]

The primary task of the emergency clinician is to distinguish syncope as a presentation of ongoing, life-threatening disease (such as subarachnoid hemorrhage, pulmonary embolism, and hypovolemia secondary to bleeding) from syncope that results from benign causes (such as vasovagal syncope), and from high-risk causes that may have resolved at the time of the ED visit (particularly arrhythmias).[5] The most effective tools for evaluating syncope remain the history, physical examination, and electrocardiogram (ECG), as advanced studies have shown vanishingly low yield for most patients with normal examinations and ECGs.[6-11] The use of structured syncope evaluation pathways may result in decreased utilization and increased yield.[12-14] Several clinical decision rules exist, but their utility has been questioned.[15,16]

This issue of *Emergency Medicine Practice* discusses the best available evidence to identify the patients with syncope who may be safely discharged with limited testing and which patients may benefit from further investigation, including hospitalization.

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Critical Appraisal of the Literature

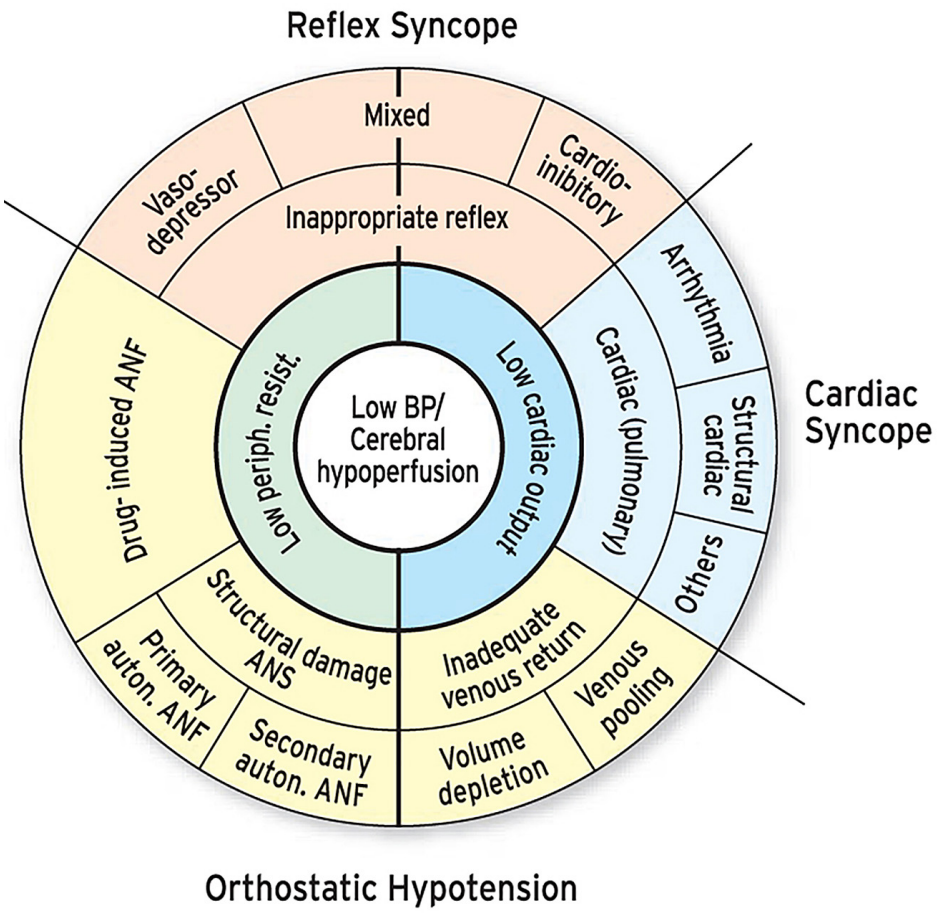
A literature search from January 1970 to December 2020 was performed in PubMed, Embase®, the Cochrane Database of Systematic Reviews, and the Cochrane Central Register of Controlled Trials, using MeSH terms including *syncope, death, hospitalization, risk assessment,* and *emergency.* The intent of the literature search was to identify studies relevant to the epidemiology, ED evaluation, and risk stratification of patients presenting with syncope. A total of 1288 abstracts were screened, and 380 full-text studies reviewed for inclusion, with 117 studies included. Practice guidelines from professional societies including the American College of Emergency Physicians (ACEP), the European Society of Cardiology (ESC), and the American Heart Association (AHA) were reviewed.

Although there are numerous studies examining syncope, many are database reviews or retrospective reviews. The definitions used for syncope and its categorizations vary considerably and, between studies, the populations are often quite heterogeneous. Risk-stratification tools often perform poorly in subsequent validation studies.[17-19] Most prospective studies have small sample sizes, which is problematic, as the low rate of serious adverse events makes it challenging to detect significant relationships reliably. Overall, the syncope literature consists primarily of observational studies and database studies, which are assigned a low quality of evidence. The plethora of clinical decision rules underscore the challenge associated with distilling such a varied presentation and population into a concise set of recommendations.

Etiology and Pathophysiology

Syncope represents the final common pathway of a number of conditions causing hypoperfusion of the brain. This hypoperfusion may result from decreased peripheral vascular resistance, decreased cardiac output, or both, and is commonly divided into 3 classifications: (1) neurally mediated (reflex) syncope, (2) orthostatic hypotension, and (3) cardiac syncope. **(See Figure 1.)** These categories can be subdivided, as noted in **Table 1, page 4**. Although no cause is identified in the majority of patients, neurally mediated syncope is the most common cause of syncope identified, followed by orthostatic syncope, and then cardiac syncope.[20] Syncope is most commonly a benign diagnosis, with a review of 22,189 patients finding a 30-day risk of death in patients aged <60 years without a history of heart failure to be 0.2%.[21] A meta-analysis of 43,315 patients found a 4.4% risk of death (95% confidence interval; 3.1%-5.1%) for all ED patients with syncope, with 1.1% due to a cardiovascular etiology. Palpitations preceding syncope, exertional syncope, and a history of heart disease were the most powerful predictors of adverse outcomes.[22]

Figure 1. The Physiologic Basis of Syncope



Abbreviations: ANF, autonomic failure; ANS, autonomic nervous system; BP, blood pressure.

Brignole M, Moya A, de Lange FJ, et al, 2018 ESC Guidelines for the diagnosis and management of syncope, *European Heart Journal* 2018; 39 (21): 1883 -1948 doi:10.1093/eurheartj/ehy037. Reprinted by permission of Oxford University Press on behalf of the European Society of Cardiology

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Neurally Mediated Syncope

Neurally mediated syncope may result from inappropriate bradycardia, decreased vasomotor tone, or both, and is more common in those younger than 40 years. Younger people are more likely to have typical prodromal symptoms, such as nausea and sweating, than older adults.[23] Vasovagal syncope is frequently triggered by pain or intense emotions and, in susceptible individuals, can be provoked by medical procedures and the sight of blood. Situational syncope is associated with increased vagal tone secondary to particular actions, including Valsalva maneuvers, following exercise, and occasionally gastrointestinal stimulation such as swallowing or defecation. Carotid sinus syncope may be induced from pressure on the carotid sinus such as head turning or shaving. Neurally mediated syncope is commonly recurrent, and is associated with a good long-term prognosis.[24]

Orthostatic Hypotension-Mediated Syncope

Orthostatic hypotension is defined as a fall in the systolic blood pressure of >20 mm Hg upon standing, or symptoms such as dizziness. It results from volume depletion or autonomic failure, which is the inability of the sympathetic nervous system to increase vascular tone with changes in posture. Drug-related hypotension is the most frequent cause, although patients with orthostatic hypotension are usually older and have multiple comorbidities.[25] Orthostasis may cause syncope by itself, or it may be incidentally present in other, more concerning causes. One study found no difference in 30-day serious outcomes between elderly patients with normal orthostatic vital signs and those with abnormal orthostatic vital signs.[26]

Cardiac Syncope

Cardiac syncope carries the highest morbidity of all causes, and patients with underlying cardiac disease who experience syncope have higher all-cause rates of mortality.[27] Cardiac syncope may result from absent or ineffective contractions due to obstruction of blood flow (as with pulmonary embolism, aortic stenosis, and aortic dissection), or it may result from dysrhythmias. Dysrhythmias are the most common causes of cardiac syncope, and may be challenging to diagnose, as they will most likely have resolved by the time of ED evaluation. A careful history and review of the ECG may provide some clues to an underlying etiology. Obstructive or structural causes of cardiac syncope will typically persist through the ED evaluation and are more easily identified.

Table 1. Classifications of Syncope, by Cause

Neurally Mediated (Reflex) Causes

- **Vasovagal**
  - Fear
  - Pain
  - Medical procedures/instrumentation (eg, intravenous line insertion, blood draw)
  - Noxious stimuli (eg, sight of blood)
  - Valsalva maneuver
  - Breath-holding
  - Prolonged standing
- **Situational**
  - Cough
  - Micturition
  - Gastrointestinal stimulation (swallowing, defecation)
  - Post exercise
- **Carotid Sinus Stimulation/Hypersensitivity**

Orthostatic Hypotension-Mediated Causes

- **Volume Depletion**
  - Vomiting/diarrhea
  - Dehydration
  - Drug-induced (eg, diuretics, vasodilators)
  - Hemorrhage
- **Distributive Shock (Sepsis, Anaphylaxis)**
- **Autonomic Failure**
  - Primary autonomic failure (eg, multiple system atrophy, Parkinson disease)
  - Secondary autonomic failure (eg, diabetes, amyloidosis)
  - Drug-induced (eg, sedatives, antihypertensives)

Cardiac Causes

- **Dysrhythmias**
  - Bradycardias (sinus node dysfunction, high-grade atrioventricular block, sick sinus)
  - Tachycardias (ventricular tachycardia, supraventricular tachycardia, torsades)
  - Pacemaker malfunction
  - Inherited cardiac disease/channelopathies (Brugada syndrome, long QT syndrome)
- **Valvular Disease**
  - Severe aortic stenosis
  - Valve rupture/prosthetic valve dysfunction
- **Structural Disease**
  - Aortic dissection
  - Cardiomyopathy, hypertrophic cardiomyopathy
  - Heart failure
  - Cardiac tamponade
  - Ischemic heart disease
- **Other Cardiovascular Causes**
  - Pulmonary embolism
  - Pulmonary hypertension
  - Subclavian steal syndrome

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Differential Diagnosis

The initial focus of evaluation in a patient presenting with syncope must be on the identification or exclusion of immediately life-threatening conditions that may present with a TLOC. Many serious conditions can result in a loss of consciousness, but they typically do not resolve spontaneously with return to baseline status by the time of ED evaluation. Any patient with neurologic deficit, headache, chest pain, abdominal pain, or persistent abnormal vital signs should not be diagnosed with syncope unless all more serious diagnoses are excluded. Patients determined to have a nonsyncopal etiology of their loss of consciousness should be evaluated as appropriate for that etiology. **See Table 2** for clinical findings that suggest a life-threatening etiology.

In patients for whom a life-threatening etiology is not apparent, the history is most helpful in distinguishing between syncope, seizure, psychogenic causes of TLOC, and rare causes of TLOC such as posterior circulation transient ischemic attack. Metabolic causes such as hypoglycemia and certain toxins or overdoses may result in TLOC, but they uncommonly resolve spontaneously. Pharmacological treatments may cause orthostatic hypotension or prolonged QT intervals. Forty-five percent of patients with unexplained syncope may carry a diagnosis of past or current substance abuse, and men with a history of psychiatric illness are more likely to have unexplained syncope.[28,29] Patients with psychiatric disorders are susceptible to conditions that may mimic syncope. **See Table 3** for a list of syncope mimics.

Table 2. Clinical Characteristics of Immediately Life-Threatening Conditions That May Present With Syncope

Life-Threatening Condition	Clinical Characteristic
Subarachnoid hemorrhage	• Sudden-onset, severe headache • Neurologic deficit
Cerebrovascular accident	• Neurologic deficit
Acute myocardial infarction	• Chest pain • Shortness of breath • Nausea, diaphoresis
Aortic dissection	• Sudden-onset, tearing chest pain • Neurologic or pulse deficit • Pain radiating to the back
Pulmonary embolism	• Tachycardia, tachypnea • Shortness of breath, chest pain • Hypoxia
Abdominal aortic aneurysm	• Abdominal pain • Pain radiating to the back • Neurologic or pulse deficit
Gastrointestinal bleeding	• Tachycardia • Hematemesis, melena
Ectopic pregnancy	• Abdominal or pelvic pain • Vaginal bleeding
Sepsis	• Fever, tachycardia, or hypotension • Clinical signs of infection (cough, rash, dysuria, etc)

Seizure

It may be challenging to differentiate seizure from syncope, as syncope is commonly accompanied by myoclonic convulsive activity (particularly if the patient is prevented from lying flat for some reason, eg, syncope while sitting). The most helpful feature indicating seizure is the presence of disorientation lasting more than a few seconds following the event (in witnessed events), or age <45 years (in unwitnessed events).[30,31] Features that point toward seizure include tongue laceration, head-turning, and unusual posturing. Features that indicate syncope include any presyncope (defined as autonomic activation, lightheadedness, or palpitations), loss of consciousness with prolonged standing or sitting, and diaphoresis before the spell.[32] Urinary incontinence does not distinguish between seizure and nonseizure causes of TLOC.[33] If a reliable estimate of the number of jerks is available, fewer than 10 jerks may indicate syncope, with more than 20 jerks indicating seizure.[31]

Prehospital Care

Prehospital care providers play a pivotal role in the evaluation of many patients with syncope because of their ability to obtain bystander history and their evaluation of the patient in the minutes immediately following a TLOC. In addition to standard care (including a fingerstick glucose) and assessment for trauma resulting from the syncopal episode, prehospital providers should attempt to ascertain the events preceding the loss of consciousness, its duration, any

Table 3. Syncope Mimics

- **Neurologic Causes Without Generalized Cerebral Hypoperfusion**
  - Generalized seizures
  - Complex partial seizures
  - Intracerebral hemorrhage
  - Subarachnoid hemorrhage
  - Vertebrobasilar transient ischemic attack
  - Complicated migraine
- **Psychiatric Causes**
  - Psychogenic pseudosyncope
  - Anxiety disorder
  - Cataplexy
  - Somatization disorder
- **Metabolic Disorders**
  - Hypoglycemia
  - Hypoxia
  - Hypercapnia
  - Hypocapnia (hyperventilation)
- **Intoxication**

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unusual movement during the spell, and the patient's orientation immediately following the spell. Bystanders will often focus on any movement and label the episode a "seizure;" describing the exact movements seen will be helpful for the clinician later on in confirming the ultimate diagnosis.[31]

If possible, an ECG should be acquired early on to rule out ischemia and potentially identify a transient arrhythmia or conduction abnormality. Patients for whom a life-threatening cause of syncope is suspected (eg, myocardial ischemia, hemorrhage, stroke, etc) should be preferentially transported to the nearest appropriate facility, such as a trauma center or designated stroke center.

Emergency Department Evaluation

In evaluating the patient who presents to the ED with a TLOC, the emergency clinician must first determine whether syncope is the predominant reason for the patient's presentation or whether it is a symptom of a more serious underlying condition. If the presence of an immediate threat to life is determined, then resuscitation or treatment should begin accordingly. Otherwise, the focus then shifts to accurately identifying syncope and classifying its etiology. If an etiology cannot be determined reliably in the ED, the clinician should attempt to risk-stratify the patient to determine the appropriate disposition and follow-up plan.

History

Because serious short-term outcomes of syncope are strongly associated with the etiology assigned in the ED, a thorough assessment of the events surrounding the syncopal episode is imperative to accurately diagnose the cause.[34] History must also be sought from bystanders, family members, and prehospital providers, as it is expected that the patient will not remember the event itself. Inquire specifically about activity and symptoms immediately before the event, as well as during and after the loss of consciousness, and try to elicit information such as their position (standing, sitting, or changing position; turning head), environment, prodrome, palpitations, exertion, and duration. Symptoms such as chest pain, shortness of breath, headache, and neurological deficit should be noted. Syncope without prodrome may be more likely to be caused by arrhythmias.[35]

Ask whether the patient has a history of passing out or fainting, and if they do, whether this episode differs from previous episodes. Recurrent syncope is common, but does not appear to be a significant predictor of mortality.[36,37] In particular, vasovagal syncope often has known triggers.[38] Inquire about any previous treatment or evaluation, including cardiac monitoring. Falls without a clear mechanism should be considered as potential syncopal events, particularly in elderly patients, as 25% to 50% of unexplained falls in this group may be related to syncope.[39-41]

The role of family history is controversial. Patients with a known family history of sudden cardiac death due to arrhythmogenic conditions such as long QT syndrome or Brugada syndrome may be at higher risk, especially if their ECG demonstrates a prolonged QTc (>460 ms) on multiple ECGs, or spontaneous type 1 Brugada morphology.[42,43] In the setting of a diagnostic type 1 Brugada ECG pattern, however, family history is not an independent predictor for future arrhythmic events.[44] Nonetheless, in the absence of an abnormal ECG, even patients with a positive family history may not be at increased risk.[44] Interestingly, cases of sudden cardiac death among younger people may be missed because they are classified as, for example, a drowning or motor vehicle crash.

The term *presyncope* may be used to describe prodromal symptoms such as lightheadedness, sweating, nausea, or palpitations without a complete loss of consciousness. The outcomes of patients presenting with presyncope versus syncope do not appear to differ significantly, and the absence of a complete loss of consciousness should not alter the ED evaluation.[45,46]

Inquire about known medical conditions, and all medications, herbal supplements, and other substances the patient may be taking. Pay particular attention to any new or recently changed medications, as almost 40% of cases of orthostatic hypotension syncope may be drug-induced.[25] Patients with a history of cardiac disease, particularly heart failure, are at higher risk for adverse outcomes.[47] **See Table 4, page 7** for historical features that may indicate a particular category of syncope.

Physical Examination

The primary value of the physical examination is to assess for findings that indicate a life-threatening cause of syncope, such as tachycardia, bradycardia, hypotension, tachypnea, hypoxia, or neurologic deficits. In most patients for whom syncope is the predominant presentation, the physical examination and vital signs will be normal. Look for evidence of hypovolemia or hypoperfusion, which may indicate hemorrhage or sepsis, and examine the abdomen to assess for tenderness or a pulsatile mass, which could indicate an abdominal aortic aneurysm. Although transient hypotension is expected with syncope, persistent, unexplained low blood pressure in the ED indicates a more serious etiology. If possible, consider obtaining orthostatic vital signs. Perform a careful neurologic examination to evaluate for subtle deficits that may indicate stroke.

Syncope is a frequent cause of significant injuries, with one study finding 6% of syncope patients experiencing fractures or motor vehicle crashes, and 29% incurring minor injuries such as lacerations and bruises.[48] Particularly in older patients, a thorough

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physical examination should be performed to look for any evidence of trauma.

The cardiopulmonary examination is particularly important. Listen for irregular rhythms and murmurs and, if present, inquire as to any previous echocardiography or known diagnoses. Look for evidence of obstruction, such as distended neck veins or muffled heart sounds, and for evidence of heart failure, such as pulmonary rales and pedal edema.

In patients aged >40 years who have undiagnosed syncope after the initial evaluation that has included a history, physical examination, and ECG, both the AHA and the ESC recommend performing carotid sinus massage.[5,49] Carotid sinus hypersensitivity is diagnosed when carotid sinus massage results in syncope, asystole >3 seconds, or atrioventricular block, or there is a drop of at least 50 mm Hg in systolic blood pressure. Carotid sinus massage should be avoided in certain patients **(see Table 5)** and should be conducted with appropriate monitoring and resuscitation equipment readily available. Although carotid sinus massage may be diagnostic in up to 60% of patients (increasing with advanced age), approximately 0.1% of patients may have neurologic complications from the procedure, and careful patient selection is important.[50,51]

Table 4. Historical Features Suggesting Etiology of Syncope

- **Neurally Mediated Syncope**
  - Long history of syncope, especially if beginning before age 40
  - Triggered by pain, medical procedure, environmental stimuli (sight, sound, smell)
  - Occurs after prolonged standing
  - Prodrome of nausea, sweating, pallor, or flushing; "tunnel vision"
  - Associated with crowds or hot places
  - After exercise or exertion
  - During a meal
  - During or after micturition, coughing, swallowing, or defecation
  - Associated with head-turning or pressure on carotid sinus (shaving, tight collars)
- **Orthostatic Hypotension-Mediated Syncope**
  - Shortly after standing
  - Prolonged standing or standing after exertion
  - Postprandial hypotension
  - Associated with initiation or increased dose of certain medications
  - History of autonomic neuropathy
- **Cardiac Syncope**
  - During exertion or while supine
  - Palpitations preceding syncope or no prodrome
  - Family history of sudden cardiac death at a young age
  - Known structural heart disease or coronary artery disease

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Table 5. Contraindications to Performing Carotid Sinus Massage

- Among patients at high risk for stroke:
  - Transient ischemic attack or stroke within 3 months
  - Known clinical carotid stenosis
  - Carotid bruit
  - Peripheral artery disease
  - Coronary artery disease
- Complications from previous massage
- Myocardial infarction in the previous 3 months
- History of ventricular arrhythmias

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Diagnostic Studies

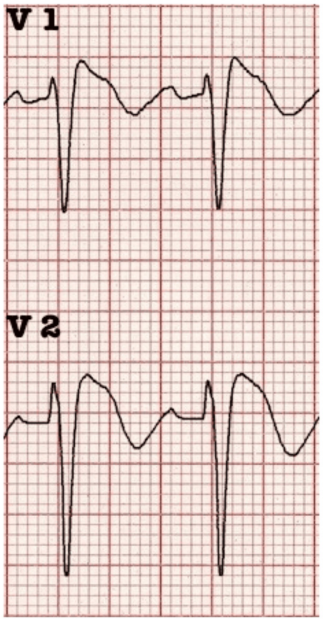
Unless indicated by the history and physical examination, very few diagnostic studies aid in the evaluation of syncope. Imaging (eg, chest x-ray, head computed tomography [CT]), in particular, has a very low yield in determining the cause of syncope and should not be ordered unless it is independently indicated.

Electrocardiogram

An ECG should be obtained for all patients with syncope. Review the ECG carefully to determine the underlying rhythm and rate, then look for conduction abnormalities such as pre-excitation or bundle branch block, ischemia and left ventricular hypertrophy, and rare findings such as type 1 Brugada morphology **(see Figure 2)** or epsilon waves associated with arrhythmogenic right ventricular cardiomyopathy **(see Figure 3, page 8)**.

Certain specific abnormalities appear to be helpful in risk stratifying patients with syncope.[52] The ECG is more likely to be diagnostic in patients aged >40 years, and several findings are associated

Figure 2. Type 1 Brugada Syndrome



Note the coved ST elevation of over 2 mm followed by a negative T wave.
Source: https://litfl.com/brugada-syndrome-ecg-library/
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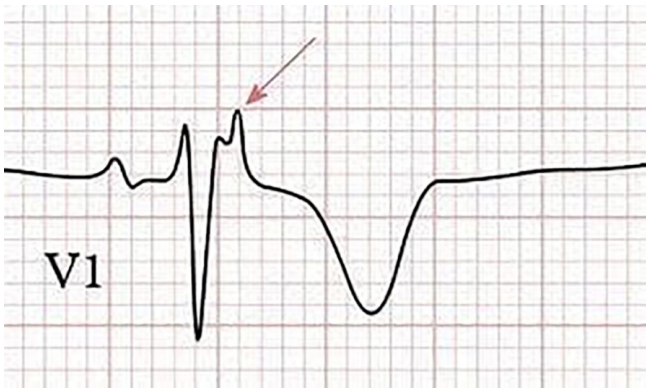
with serious cardiac arrhythmias within 30 days.[53,54] **(See Table 6.)** A 2014 study found atrial fibrillation, intraventricular conduction disturbances, left ventricular hypertrophy, and ventricular pacing to be independent predictors of mortality.[55] Other ECG findings that suggest a cardiac cause of syncope include Mobitz I atrioventricular block, inappropriate sinus bradycardia, early repolarization, and left ventricular hypertrophy, particularly if associated with "dagger" Q waves suggesting hypertrophic cardiomyopathy.[7,56] **(See Figure 4.)**

Patients with syncope should be placed on continuous telemetry while in the ED. In low-risk patients, half of arrhythmic causes of syncope are detected within 2 hours of ED arrival, and within 6 hours for medium-risk and high-risk patients.[57] Early referral for ambulatory monitoring may be helpful, with a higher diagnostic yield for patients in whom monitoring is initiated within 15 days of the event.[58]

Laboratory Testing

Laboratory tests are of low yield in patients with syncope unless the history or physical examination indicate a serious underlying disease process. For patients without a clear diagnosis of a low-risk syncope (such as vasovagal syncope), consider obtaining a complete blood cell count (CBC), glucose level, electrolyte levels, and renal function tests, as well as a pregnancy test in women of child-bearing age. A 2018 meta-analysis found elevated blood urea nitrogen and abnormal troponin to be predictive of short-term serious events.[59] Several risk-stratification tools include anemia as a high-risk finding, although patients with life-threatening causes of syncope such as ruptured abdominal aortic aneurysm or ectopic pregnancy may have initially normal blood counts, and the CBC should not be used to rule out these causes. An elevated serum lactate measurement obtained within 2 hours of the event is superior to creatine kinase measurement in distinguishing syncope from seizure, with a positive likelihood ratio of 5.8.[60]

Figure 3. Epsilon Wave

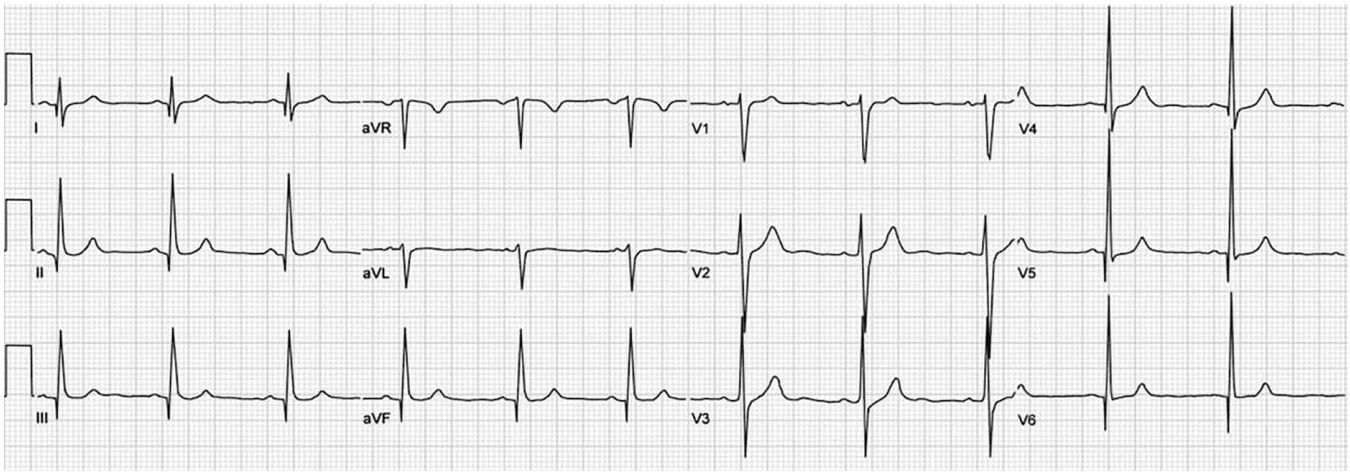


The small positive deflection after the QRS (arrow), with inverted T waves in the precordial leads suggests arrhythmia as the cause of syncope secondary to arrythmogenic right ventricular cardiomyopathy.
Source: https://litfl.com/epsilon-wave-ecg-library/
Used with permission of Life in the Fastlane, https://litfl.com/

Table 6. Electrocardiogram Findings Associated With Serious Cardiac Arrhythmias Within 30 Days

- Non–sinus rhythm
- Multiple premature ventricular contractions
- Short PR interval
- First-degree atrioventricular block
- Complete left bundle branch block
- Findings consistent with acute or chronic ischemia (Q waves, T-wave/ST-segment changes)

Figure 4. Electrocardiogram of Hypertrophic Cardiomyopathy



The deep, narrow Q waves in the inferior and lateral leads suggest hypertrophic cardiomyopathy.
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The role of cardiac biomarkers in the risk stratification of syncope is evolving, with studies performed after the widespread adoption of high-sensitivity cardiac troponin (hs-cTnT) testing generally more likely to show utility in risk stratification. Elevated hs-cTnT and N-terminal pro B-type natriuretic peptide (NT-proBNP) are independent predictors of 30-day mortality, although this may represent a significant fraction of patients who would already be considered to be high-risk due to underlying illness.[61-63] An elevated BNP may confer an 8-fold increase in the risk for serious outcomes, and a 6-hour increase in BNP after a syncopal episode may distinguish ventricular fibrillation or ventricular tachycardia from vasovagal syndrome as the cause.[64,65] In patients with undiagnosed syncope after the history, physical examination, and ECG, is it reasonable to obtain a BNP or proBNP and hs-cTnT to aid in recognition of cardiac syncope and risk stratification.[66-68]

Pulmonary embolism is uncommon among patients presenting with syncope, and existing clinical decision rules (such as the pulmonary embolism rule-out criteria, PERC) adequately identify patients with syncope and pulmonary embolism.[69-72] Routine D-dimer measurement in patients with syncope without independent indications for evaluation of pulmonary embolism is not indicated.[73,74]

Echocardiography

Echocardiography may provide valuable information in the evaluation of a patient with syncope, particularly when the history, physical examination, or ECG raise concern for structural abnormalities such as hypertrophic obstructive cardiomyopathy or ischemia, but otherwise, it has low yield for most syncope patients.[8,75] A 2018 study found history of heart failure, coronary artery disease, abnormal ECG, and elevated hs-cTnT or NT-proBNP to be predictive of clinically significant findings on echocardiography.[76]

Imaging and Other Advanced Testing

Advanced imaging (particularly head CT) and chest x-rays are frequently obtained for syncope patients, despite their low yield and the ACEP "Choosing Wisely" recommendation to avoid head CT in asymptomatic patients with syncope, minimal trauma, and a normal neurologic examination.[77] In the absence of neurological symptoms or indications for imaging in trauma by clinical decision rules, do not obtain head CT or magnetic resonance imaging (MRI) for patients with syncope.[6,11,78] Electroencephalography and stress myocardial imaging should not be obtained unless otherwise indicated (ie, concern for seizure or evidence of myocardial ischemia, respectively).[10,79,80] Referral for electrophysiological studies may be considered, although most abnormal electrophysiology studies can be predicted, based on clinical factors such as abnormal ECG, abnormal left ventricular ejection fraction, and age.[81,82]

Treatment

If no specific diagnosis is found after initial ED evaluation, the focus of syncope evaluation will shift to risk stratification of patients into high-risk or low-risk categories. **(See Table 7, page 10.)** The wide variety of definitions for adverse events and time frames makes consensus recommendations challenging. A 2010 study of more than 35,000 patients found a 3% chance of 7-day cardiac outcomes. Factors predictive for an adverse event included age >60 years, male gender, heart failure, coronary artery disease, abnormal ECG, or structural heart disease.[83] A 2018 meta-analysis found that among patients with noncardiac or unexplained syncope, older, hypertensive, and diabetic patients had higher all-cause mortality.[84]

Clinical Risk-Stratification Tools

Risk-stratification tools should be applied only to patients for whom no apparent cause of syncope has been determined after the history, physical examination, and ECG have been performed. Overall, risk-stratification tools do not appear to perform better than unstructured clinical judgment in predicting adverse events, and their application does not appear to reduce admission rates.[15,85,86] Commonalities across several clinical tools include the categorization of patients with concerning cardiac symptoms or histories, abnormal ECGs or biomarkers, or anemia as being "high risk;" and those with vasovagal syncope as being "low risk." **See Table 8, page 11** for a comparison of features of several risk-stratification tools, which are summarized following:

- **The OESIL** (Osservatorio Epidemiologica sulla Sincope nel Lazio) risk score was derived in 2003 to predict 1-year mortality in patients presenting with syncope. Mortality increased from 9% for patients with 0 points to 57.1% for patients meeting all 4 criteria (4 points).[87]
- **The San Francisco Syncope Rule (SFSR)** considers the presence of any of 5 features to indicate high risk. It was derived in 2004 to predict serious outcomes within 7 days and was reported to be 96% sensitive and 62% specific in derivation and 98% in prospective validation.[88,89] Subsequent studies looked at 30-day outcomes. External validation studies found the sensitivity as low as 76%, and strict application of the rule may actually increase rather than decrease admissions.[16,90,91]
- **The Boston Syncope Criteria (BSC)** consist of 25 features divided into 7 categories; any single feature is considered to indicate high risk. It was derived in 2007 and found to be 97% sensitive and 62% specific for adverse outcomes at 1 month.[56] The lack of external validation and large number of criteria may limit its use in the ED setting.

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Table 7. Low-Risk and High-Risk Features of Syncope, European Society of Cardiology[5]

SYNCOPAL EVENT	
Low-Risk	
• Associated with prodrome typical of reflex syncope (eg, light-headedness, feeling of warmth, sweating, nausea, vomiting) • After sudden unexpected unpleasant sight, sound, smell, or pain • After prolonged standing or crowded, hot places • During a meal or postprandial	• Triggered by cough, defecation, or micturition • With head rotation or pressure on carotid sinus (eg, tumor, shaving, tight collars) • Standing from supine/sitting position
High-Risk	
Major	
• New-onset chest discomfort, breathlessness, abdominal pain, or headache	• Syncope during exertion or when supine • Sudden-onset palpitation immediately followed by syncope
Minor (high risk only if associated with structural heart disease or abnormal ECG)	
• No warning symptoms or short (<10 s) prodrome • Family history of sudden cardiac death at young age	• Syncope in the sitting position
PAST MEDICAL HISTORY	
Low-Risk	
• Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode • Absence of structural heart disease	
High-Risk	
Major	
• Severe structural or coronary artery disease (heart failure, low left ventricular ejection fraction, or previous myocardial infarction)	
PHYSICAL EXAMINATION	
Low-Risk	
• Normal examination	
High-Risk	
Major	
• Unexplained systolic BP in the ED <90 mm Hg • Suggestion of gastrointestinal bleed on rectal examination	• Persistent bradycardia (<40 beats/min) in awake state and in absence of physical training • Undiagnosed systolic murmur
ECG[a]	
Low-Risk	
• Normal ECG	
High-Risk	
Major	**Minor** (high-risk only if history consistent with arrhythmic syncope)
• ECG changes consistent with acute ischemia • Mobitz II second- and third-degree AV block • Slow AF (<40 beats/min) • Persistent sinus bradycardia (<40 beats/min) or repetitive sinoatrial block or sinus pauses >3 sec in awake state and in absence of physical training • Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy, or Q waves consistent with ischemic heart disease or cardiomyopathy • Sustained and nonsustained VT • Dysfunction of an implantable cardiac device (pacemaker or ICD) • Type 1 Brugada pattern • ST-segment elevation with type 1 morphology in leads V1-V3 (Brugada pattern) • QTc >460 ms in repeated 12-lead ECGs indicating LQTS	• Mobitz I second-degree AV block and first-degree AV block with markedly prolonged PR interval • Asymptomatic inappropriate mild sinus bradycardia (40-50 beats/min) or slow AF (40-50 beats/min) • Paroxysmal supraventricular tachycardia or AF • Pre-excited QRS complex • Short QTc Interval (≤340 ms) • Atypical Brugada patterns • Negative T waves in right precordial leads, epsilon waves suggestive of arrhythmogenic right ventricular cardiomyopathy

Abbreviations: AF, atrial fibrillation; AV, atrioventricular; BP, blood pressure; ECG, electrocardiogram; ED, emergency department; ICD, implantable cardioverter defibrillator; LQTS, long QT syndrome; VT, ventricular tachycardia.

[a]Some ECG criteria are per se diagnostic of the cause of the syncope (see recommendations: Diagnostic Criteria); in such circumstances, appropriate therapy is indicated without further investigations. We strongly suggest the use of standardized criteria to identify ECG abnormalities with the aim of precise diagnosis of ECG-defined cardiac syndromes in ED practice.

Brignole M, Moya A, de Lange FJ, et al, 2018 ESC Guidelines for the diagnosis and management of syncope, *European Heart Journal* 2018; 39 (21): 1883–1948 doi:10.1093/eurheartj/ehy037. Reprinted by permission of Oxford University Press on behalf of the European Society of Cardiology.

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- **The Risk Stratification of Syncope in the Emergency Department (ROSE) Rule** considers any of 7 features to indicate high risk. It was derived in 2010 and includes measurement of BNP and hemoglobin, along with occult blood on rectal examination, to risk-stratify patients for adverse outcomes at 1 month. The initial study found a negative predictive value of 98.5%, with a sensitivity of 87% and specificity of 65.5%. No large validation studies have been performed on this tool.[92] The ROSE rule does not predict mortality at 1 year.[93]

Table 8. Clinical Decision Tools Used in Syncope Risk Stratification

Tool	Criteria	Score Cutoff	Outcome
OESIL	• Age >65: 1 point • Cardiovascular disease: 1 point • No prodrome: 1 point • Abnormal ECG: 1 point MDCalc: www.mdcalc.com/oesil-score-syncope	Score of ≥2 = high risk	Mortality at 1 year
SFSR	**C** – History of **C**ongestive heart failure **H** – **H**ematocrit <30% **E** – Abnormal **E**CG **S** – Shortness of breath **S** – SBP <90 mm Hg at triage MDCalc: www.mdcalc.com/san-francisco-syncope-rule	Any feature = high risk	Serious outcomes at 7 days
BSC	• Signs/symptoms of ACS • Signs of conduction disease • Worrisome cardiac history • Family history of sudden cardiac death • Volume depletion • Persistent abnormal vital signs in the ED • Central nervous system event	25 criteria in categories, any feature = high risk	Serious outcomes at 30 days
ROSE	• BNP ≥300 pg/mL • Bradycardia <50 beats/min in ED or prehospital • Rectal examination showing occult blood • Anemia: hemoglobin <9 g/dL • Chest pain • ECG showing Q wave (not in III) • Oxygen saturation ≤94% on room air	Any feature = high risk	Serious outcomes at 7 days, 1 month, and 3 months
EGSYS	• Palpitations preceding syncope: 4 points • Heart disease or abnormal ECG or both: 3 points • Exertional syncope: 3 points • Supine syncope: 2 points • Precipitating or predisposing factors: -1 point • Autonomic prodrome: -1 point MDCalc: www.mdcalc.com/egsys-evaluation-guidelines-syncope-study-score-syncope	Total score of ≥3 = high risk	Serious outcomes at 21-24 months
CSRS	• Setting suggests vasovagal syncope: -1 point • History of heart disease: +1 point • Any SBP <90 mm Hg or >180 mm Hg: +2 points • Elevated troponin: +2 points • Abnormal QRS axis: +1 point • QRS duration >130 ms: +1 point • QTc >480 ms: +2 points • Diagnosis of vasovagal syncope in ED: - 2 points • Diagnosis of cardiac syncope in ED: +2 points MDCalc: www.mdcalc.com/canadian-syncope-risk-score	Score of >-2	Death or serious adverse event at 30 days
FAINT	**F:** Heart **F**ailure: 1 point **A:** History of cardiac **A**rrhythmia: 1 point **I:** Initial abnormal ECG: 1 point **N:** Elevated pro-B**N**P: 2 points **T:** Elevated high sensitivity-**T**roponin: 1 point	Score of ≥1 = high risk	Serious outcomes at 30 days

Abbreviations, ACS, acute coronary syndromes; BNP, brain natriuretic peptide; BSC, Boston Syncope Criteria; CSRS, Canadian Syncope Risk Score; ECG, electrocardiogram; ED, emergency department; EGSYS, The Evaluation of Guidelines in Syncope Study; OESIL, Osservatorio Epidemiologica sulla Sincope nel Lazio; ROSE, Risk Stratification of Syncope in the Emergency Department Rule; SBP, systolic blood pressure; SFSR, The San Francisco Syncope Rule.

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- **The Evaluation of Guidelines in Syncope Study (EGSYS)** assigns points to clinical features to predict cardiac syncope. A score of ≥3 is considered to indicate high risk. It was derived in 2008 and reported to be 95% sensitive for predicting death at 21 to 24 months,[94] but a 2012 external validation found the sensitivity to be 80%.[95]
- **The Canadian Syncope Risk Score (CSRS)** was derived in 2016 and assigns positive or negative points to clinical variables to identify the risk for serious outcomes at 30 days. A score >-2 is 99% sensitive for serious outcomes. The rule has been validated in a large multicenter trial, and the rate of adverse outcomes increases substantially for higher scores.[96,97]
- **The FAINT score** attempts to identify risk of death and serious cardiac outcomes at 30 days in adults aged >60 years with syncope or presyncope. It was derived in 2020 and has a sensitivity of 96.7% for a score of ≥1, with slightly improved performance over physician judgment, but it has not yet been validated.[98]

Online tools for risk stratification for syncope are available at MDCalc:

- OESIL Score for Syncope: www.mdcalc.com/oesil-score-syncope
- San Francisco Syncope Rule: www.mdcalc.com/san-francisco-syncope-rule
- EGSYS Score for Syncope: www.mdcalc.com/egsys-evaluation-guidelines-syncope-study-score-syncope
- Canadian Syncope Risk Score: www.mdcalc.com/canadian-syncope-risk-score

Guidelines

Comprehensive, updated guidelines were released in 2017 by the AHA/American College of Cardiology (ACC)[49] and in 2018 by the ESC.[5] Both guidelines support the use of a detailed history, physical examination, and ECG as the initial evaluation, and then risk stratification rather than diagnosis in patients for whom the etiology remains uncertain. The use of clinical decision tools is considered reasonable, although outcomes correlate strongly with the etiology assigned in the ED, and clinical decision tools should not replace physician judgment.[34,99]

The ACEP 2007 Clinical Policy on syncope[100] has not been updated since its expiration, but ACEP has endorsed the 2017 ACC/AHA/Heart Rhythm Society (HRS) guidelines. A 2019 study found no significant changes in practice patterns associated with the release of the 2007 clinical policy.[77]

Special Populations

Pediatric Patients

The approach to syncope in children mirrors that of adults. The history, physical examination, and ECG are the most useful components of a syncope evaluation in children, and the vast majority of children have a benign etiology for syncope. Neurally mediated syncope accounts for 80% of cases of children with syncope evaluated in the ED, and the majority of cases of cardiac syncope may be diagnosed by history, physical examination, and ECG.[101] Family history, exertional syncope, syncope without prodrome, and syncope while supine may suggest a cardiac cause, and syncope associated with a loud noise should prompt evaluation for long QT syndrome.[102] Pediatric patients with syncope often receive extensive evaluation, including head CT (58%, in one study) and laboratory testing, with very low yield, and 10% are admitted, with very few ultimately receiving a diagnosis of cardiac syncope.[103]

For more information on syncope in children, see the April 2017 issue of *Pediatric Emergency Medicine Practice*, "Syncope in Pediatric Patients: A Practical Approach to Differential Diagnosis and Management in the Emergency Department," at: www.ebmedicine.net/pedsyncope

Elderly Patients

The evaluation of syncope in older adults is particularly challenging, as many do not present with syncope, but rather with unexplained falls. Older adults are more likely to have underlying high-risk medical conditions, yet may still have benign causes of syncope. One-fourth of adults aged >50 years who present to the ED with a fall have symptoms suggestive of syncope or unexplained fall.[40] A 2016 study found almost half of older adults with dementia referred for unexplained falls received a diagnosis of syncope,[41] and neurally mediated syncope is common in this age group.[39] QTc prolongation may occur in 25% of elderly patients, and increasing age or QTc >500 ms may predict 30-day and 1-year mortality.[104,105]

Controversies

Admission of Elderly Patients

Older adults presenting with syncope often have multiple risk factors and, overall, have worse outcomes than younger patients. Nonetheless, a 2019 study of 2492 patients found that, for older adults with unexplained syncope and no serious ED diagnosis, hospitalization did not improve 30-day adverse outcomes.[106] This supports earlier studies that found that age >65 years alone and unexplained syncope were not independent predictors of mortality in elderly patients.[107,108] Hospitalization may be associ-

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ated with a greater likelihood of identifying a serious diagnosis, but it does not appear to be associated with improved mortality, which is related primarily to comorbidities.[109,110]

Orthostatic Vital Signs

Many guidelines recommend obtaining formal orthostatic vital signs on patients presenting with syncope. Although reasonable, there is likely some overlap between patients with asymptomatic changes in blood pressure and those with true orthostatic hypotension-mediated syncope, and the presence of orthostatic hypotension does not preclude more serious causes of syncope in the elderly.[111,112] Identifying orthostasis may be beneficial to allow for modification of risk factors (such as medications) that may increase the likelihood of falls, but it should not be used as the sole factor in risk stratification.

Disposition

High-Risk Patients

Patients with an identified life-threatening diagnosis should receive treatment for that specific diagnosis, and they need not undergo syncope risk stratification. Patients with syncope that remains unexplained after the history, physical examination, ECG, and initial testing should be risk-stratified using appropriate clinical features, and those with any high-risk features should be considered for admission to the hospital or an observation unit, if available. Patients who do not require active treatment are generally appropriate for observation with monitoring, even with high-risk features.

Intermediate-Risk Patients

For patients without high-risk or low-risk features, a structured ED management pathway may allow for safe management with fewer hospital admissions.[113] Intermediate-risk factors include age ≥50 years, prior history of cardiac disease, cardiac device without evidence of dysfunction or concerning ECG findings, family history of sudden cardiac death, and symptoms not consistent with reflex-mediated syncope.[49] Patients with risk factors by past medical history (such as coronary artery disease or heart failure) but with a benign etiology of the syncopal event identified in the ED (such as dehydration) may be discharged safely.[114] Engage in shared decision-making regarding hospitalization versus outpatient follow-up, and ensure close follow-up, as many patients in this category may benefit from further testing, including tilt-table testing, implantable loop recorder, electrophysiology evaluation, or other continuous ambulatory electrocardiographic monitoring.[115] Refer older adults with unexplained falls or syncope without other risk factors to their primary care provider for further evaluation.

Low-Risk Patients

Low-risk patients, even those with unexplained syncope, should generally be discharged. Follow-up may be appropriate for patients with recurrent syncope or those without clearly benign causes. Even among patients with low-risk syncope, approximately 9% may have recurrence within 6 months, with 3 or more lifetime incidents most predictive of future recurrence.[116] Be familiar with state laws regarding release for driving, and consider referral before return to work for commercial drivers or others with a high risk for injury if syncope recurs.[117]

Summary

Syncope is a common, usually benign presentation resulting from transient cerebral hypoperfusion. It does not require extensive investigation in the ED or hospital admission unless there are indications for a life-threatening process or certain high-risk features. An appropriate, focused history, physical examination and ECG are the most important tools in identifying the cause of syncope. For patients in whom the cause is unclear after a careful history, physical examination, and ECG have been performed, laboratory testing may be helpful to identify the very small proportion of patients who may benefit from admission or evaluation in a syncope unit. Investigations not otherwise indicated by the history and physical examination, especially advanced neurological testing such as electroencephalogram or neuroimaging, have very low yield and should generally be avoided.

Society guidelines and multiple clinical decision tools have attempted, with some success, to codify a robust mechanism for identifying the subset of patients with syncope who are at high risk for mortality and other adverse outcomes. The variability in outcomes and inclusion criteria limit their inclusion in systematic reviews or meta-analyses, and the small proportion of adverse outcomes among all patients presenting with syncope requires large sample sizes that are difficult to generate. However, certain features commonly included in risk-stratification scores, such as cardiac disease, abnormal ECG, history or signs of heart failure, and possibly elevated biomarkers, may be associated with increased risk for short-term mortality and other adverse events.

Time- and Cost-Effective Strategies

- Obtain an ECG on all syncope patients, even those thought to have vasovagal syncope, and a pregnancy test in all women of child-bearing age. Otherwise, limit testing and imaging in patients for whom there is no concern for immediately life-threatening conditions such as myocardial infarction, aortic dissection, pulmonary embolism, etc.

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Case Conclusions

CASE 1

For the 16-year-old girl who was referred to the ED for a head CT after passing out in a doctor's office…

After you reassured her parents that a head CT was not indicated, given that there was no head trauma and she had a normal neurologic exam, you and the parents agreed to observe the 16-year-old girl in the ED for a few hours. Within 2 hours, she was eager to go home, and the parents were grateful to have avoided unnecessary radiation exposure.

CASE 2

For the 40-year-old man who presented by EMS after what bystanders described as a "seizure" while sitting in a chair…

You noted that the man had no tongue trauma, and there was no reported postictal period, so your concern for seizure was very low. However, the tachycardia, hypoxia, and tachypnea with borderline systolic blood pressure after his surgery 3 weeks earlier raised your concern for pulmonary embolism. You obtained an ECG, which showed an incomplete right bundle branch block, and point-of-care ultrasound showed an enlarged right ventricle, which led you to initiate heparin, pending a confirmatory CT. Submassive pulmonary embolism was diagnosed, and the patient was admitted to the ICU for thrombolytic therapy.

CASE 3

For the 80-year-old woman who presented to the ED after a fall…

The woman's unexplained fall and comorbidities led you to obtain an ECG, which showed a QTc of 500 ms. You placed her on telemetry, and shortly afterward, it captured an episode of nonsustained ventricular tachycardia. She was admitted to a monitored bed for continuous telemetry and cardiology consult, and she received an implantable cardioverter/defibrillator prior to discharge.

- Review ECGs carefully for the presence of high-risk features such as nonsinus rhythm, ectopy, conduction abnormalities including atrioventricular block and left bundle branch block, acute or chronic ischemia, abnormal intervals, and inherited conditions including hypertrophic cardiomyopathy and Brugada or long QT syndromes.
- Do not order a head CT for syncope in the absence of positive neurologic findings or suspicion for subarachnoid hemorrhage.
- Obtain orthostatic vital signs in syncope patients to identify modifiable causes of orthostatic hypotension, but be cautious of incidental orthostasis in high-risk patients.
- Consider carotid sinus massage for patients aged >40 years with unexplained syncope in the absence of contraindications.
- Do not admit low-risk patients unless injuries or other indications exist.
- Use clinical judgment, patient preference, and scientific evidence to inform disposition decisions. At this time, there is no "holy grail" of syncope risk stratification.

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References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study is included in bold type following the references, where available. The most informative references cited in this paper, as determined by the author, are noted by an asterisk (*) next to the number of the reference.

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Risk Management Pitfalls For Management of Syncope in the Emergency Department

1. **"It sounded like vasovagal syncope, so I didn't get an ECG."** Although ECGs in patients with suspected vasovagal syncope may often be normal, it is a level 1 recommendation by both the AHA and ESC to obtain an ECG in all patients with syncope. This inexpensive, noninvasive test may reveal underlying pathology that can change management dramatically.

2. **"A witness reported a seizure, so I didn't work up the patient for syncope."** Bystanders often interpret any convulsive activity as a seizure. Consider features such as tongue laceration or prolonged postictal period to diagnose syncope accurately and avoid unnecessary evaluation or restrictions on activity after discharge.

3. **"The patient had orthostasis, so I didn't think he needed admission for syncope."** The presence of orthostasis does not reliably preclude more serious causes of syncope, particularly in elderly patients. Orthostatic vital signs must be interpreted in light of the history, physical examination, and other risk factors, to avoid missing serious disease.

4. **"The patient didn't mention chest pain, so I didn't consider pulmonary embolism."** Pulmonary embolism is an uncommon cause of syncope, but should be considered in patients with unexplained persistently abnormal vital signs in the ED, especially hypotension, tachypnea, or tachycardia.

5. **"The hemoglobin was normal, so I ruled out a gastrointestinal bleed as the cause."** Acute blood loss may not be reflected in an initial hemoglobin result. Consider fecal occult blood testing in patients for whom the history or examination are suspicious for gastrointestinal bleeding (history of peptic ulcer disease, liver disease, previous gastrointestinal bleed).

6. **"There was no family history of sudden cardiac death, although he said he did have some family members drown or die in motor vehicle crashes at young ages."** The younger patient with a concerning ECG may be the first in the family to receive a diagnosis, so inquire about unexpected deaths before determining the family history is noncontributory.

7. **"A 48-hour monitor didn't show anything, so I thought arrhythmias were excluded."** Many ambulatory monitors may not be placed until weeks after the initial event, when the likelihood of capturing an arrhythmia is much lower. Failure to identify a specific arrhythmia does not exclude arrhythmia as a cause.

8. **"The ECG showed only sinus tachycardia, so I didn't do any further testing."** Abnormal vital signs that persist through the ED evaluation do not support a benign cause of syncope, and an appropriate evaluation guided by the history and physical examination should be performed to rule out a life-threatening process.

9. **"I thought her abdominal pain was from her vagal episode, so I didn't order a pregnancy test."** Nausea and abdominal pain may accompany vasovagal syncope and persist following the episode, but any patient with syncope and abdominal pain should undergo appropriate testing to exclude more serious conditions.

10. **"Since his ED evaluation was unremarkable, I released the high-rise construction worker to go back to work."** Syncope recurrence is common, but sometimes challenging to predict. Many patients with neurally mediated syncope may be safely released to return to driving, but certain high-risk occupations may require referral before returning to employment.

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1. **Which of the following most reliably distinguishes seizure from syncope?**
   a. Convulsive activity at the time of the event
   b. Serum creatine kinase results
   c. Urinary incontinence
   d. Confusion lasting for more than a few seconds after the event

2. **Which of the following symptoms is suggestive of a benign etiology of syncope?**
   a. Nausea
   b. Palpitations
   c. Shortness of breath
   d. Chest pain

3. **Regarding the evaluation of presyncope, which of the following is TRUE?**
   a. Patients with presyncope do not require an electrocardiogram (ECG) in the ED.
   b. The evaluation of presyncope in the ED should not differ from that of syncope.
   c. Imaging is more commonly required in the evaluation of presyncope.
   d. Presyncope has a more benign course and does not require admission.

4. **Which of the following underlying medical conditions is most associated with adverse outcomes in syncope?**
   a. Hypertension
   b. Seizure disorder
   c. Heart failure
   d. Carotid sinus hypersensitivity

5. **Which clinical feature of patients with Brugada syndrome is most strongly associated with adverse outcomes?**
   a. Spontaneous type 1 ECG
   b. Family history of sudden death
   c. Female sex
   d. Inducible type 2 or 3 ECG

6. **Which of the following patients should receive a head CT in the ED for syncope?**
   a. 14-year-old boy who had a syncopal episode following a blood draw
   b. 45-year-old woman with a history of heart failure and palpitations preceding syncope
   c. 67-year-old man on warfarin with syncope and a forehead contusion
   d. 22-year-old woman with syncope and a history of seizure disorder

7. **According to the 2018 European Society of Cardiology guidelines, which of the following is considered a major high-risk feature?**
   a. Unexplained low systolic blood pressure in the ED
   b. Long history of recurrent syncope
   c. Syncope with head rotation
   d. Family history of sudden cardiac death

8. **According to the Canadian Cardiovascular Society/Canadian Pediatric Cardiology Association position statement, in children, family history of syncope and sudden cardiac death, supine syncope, and syncope with loud noise suggest which of the following ECG abnormalities?**
   a. Left bundle branch block
   b. Type II second-degree atrioventricular block
   c. Sinus bradycardia
   d. Long QTc interval

9. **Regarding hospitalization for syncope, which of the following is TRUE?**
   a. The vast majority of hospitalized patients receive a diagnosis of their syncope.
   b. Among older patients for whom no serious etiology is discovered in the ED, hospitalization does not improve 30-day serious adverse events.
   c. Risk-stratification rules routinely reduce hospitalization.
   d. Hospitalization, rather than observation in a syncope unit, leads to increased diagnostic yield.

10. **Elderly patients with unexplained syncope and frequent falls should receive which of the following investigations in the ED?**
    a. Thyroid function testing
    b. Orthostatic vital signs
    c. Brain MRI
    d. Serum magnesium level

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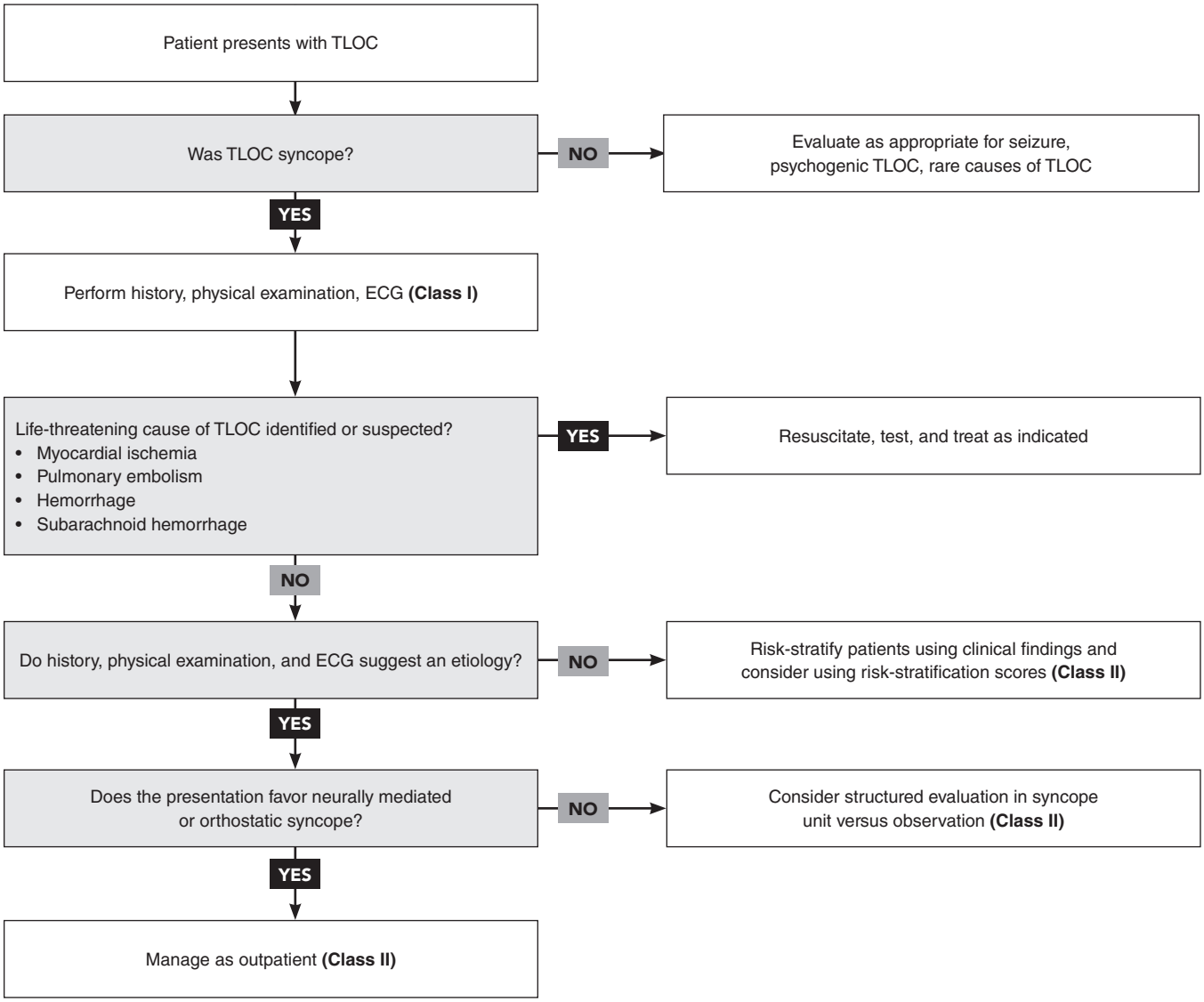
Clinical Pathway for Emergency Department Evaluation of Patients Presenting With Transient Loss of Consciousness


Patient presents with TLOC
Was TLOC syncope?
NO
Evaluate as appropriate for seizure, psychogenic TLOC, rare causes of TLOC
YES
Perform history, physical examination, ECG (Class I)
Life-threatening cause of TLOC identified or suspected?
• Myocardial ischemia
• Pulmonary embolism
• Hemorrhage
• Subarachnoid hemorrhage
YES
Resuscitate, test, and treat as indicated
NO
Do history, physical examination, and ECG suggest an etiology?
NO
Risk-stratify patients using clinical findings and consider using risk-stratification scores (Class II)
YES
Does the presentation favor neurally mediated or orthostatic syncope?
NO
Consider structured evaluation in syncope unit versus observation (Class II)
YES
Manage as outpatient (Class II)

Abbreviations: ECG, electrocardiogram; TLOC, transient loss of consciousness.

Class of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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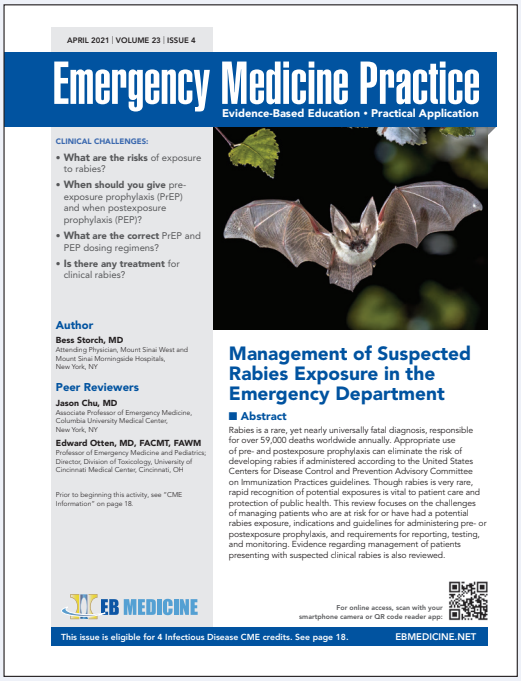
APRIL 2021 | VOLUME 23 | ISSUE 4
Emergency Medicine Practice
Evidence-Based Education • Practical Application
CLINICAL CHALLENGES:
• What are the risks of exposure to rabies?
• When should you give pre-exposure prophylaxis (PrEP) and when postexposure prophylaxis (PEP)?
• What are the correct PrEP and PEP dosing regimens?
• Is there any treatment for clinical rabies?
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Prior to beginning this activity, see "CME Information" on page 18.
Management of Suspected Rabies Exposure in the Emergency Department
Abstract
Rabies is a rare, yet nearly universally fatal diagnosis, responsible for over 59,000 deaths worldwide annually. Appropriate use of pre- and postexposure prophylaxis can eliminate the risk of developing rabies if administered according to the United States Centers for Disease Control and Prevention Advisory Committee on Immunization Practices guidelines. Though rabies is very rare, rapid recognition of potential exposures is vital to patient care and protection of public health. This review focuses on the challenges of managing patients who are at risk for or have had a potential rabies exposure, indications and guidelines for administering pre- or postexposure prophylaxis, and requirements for reporting, testing, and monitoring. Evidence regarding management of patients presenting with suspected clinical rabies is also reviewed.
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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this activity, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

CME Objectives: Upon completion of this activity, you should be able to: (1) describe clinical findings of low-risk and high-risk syncope in the emergency department; (2) identify diagnostic studies that safely and cost-effectively lead to improved risk stratification of syncope; and (3) discuss the indications for and benefit of hospitalization in syncope patients.

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QUICK READ

Points & Pearls

JUNE 2021 | VOLUME 23 | ISSUE 6

Emergency Department Management of Syncope

Points

- Syncope is a transient loss of consciousness, typically with associated loss of postural tone, followed by complete, spontaneous recovery.
- Syncope results from global cerebral hypoperfusion from decreased peripheral vascular resistance, decreased cardiac output, or both.
- There are 3 classifications: (1) neurally mediated (reflex) syncope (the most common): (2) orthostatic hypotension (second most common), and (3) cardiac syncope (the least common, but with highest morbidity). **(See Table 1, page 4.)**
- An ECG should be obtained for all patients who present with syncope, and the patient should be placed on continuous telemetry while in the ED.
- Consider a family history of sudden cardiac death, as a death perceived to be from drowning or motor vehicle crash may have actually been from unrecognized syncope.
- For elderly patients, consider falls without clear mechanism as possible syncope; 25% to 50% may be related to syncope.[39-41]
- In elderly patients, the presence of orthostatic hypotension does not preclude more serious causes of syncope.[111,112]
- Forty-five percent of patients with unexplained syncope may carry a diagnosis of past or current substance abuse; men with a history of psychiatric illness are more likely to have unexplained syncope.[28,29]
- From EMS, bystanders, or family, assess the events before and after the syncopal episode, including the position, prodrome, exertion, and duration. Note any symptoms such as palpitations, chest pain, shortness of breath, headache, or neurological deficit.
- Laboratory testing is of low yield unless the history and physical examination indicate a serious underlying disease process.
- Elevated BUN and abnormal troponin were found to be predictive of short-term serious events.[59]
- For distinguishing seizure, an elevated serum lactate within 2 hours of the event is superior to creatine kinase.[60]

Pearls

- Any patient with neurologic deficit, headache, chest pain, abdominal pain, or persistent abnormal vital signs should not be diagnosed with syncope unless all more serious diagnoses are excluded. **(See Table 2, page 5.)**
- The most helpful feature indicating seizure is disorientation lasting more than a few seconds or age <45 years.[30,31] If a reliable estimate of the number of jerks is available, <10 may indicate syncope; >20 may indicate seizure.
- If there is no apparent immediate threat to life, attempt to identify the type and etiology of the syncope; if that cannot be determined, attempt to risk-stratify the patient for high-risk or low-risk features. **(See Table 7, page 10.)**
- For patients undiagnosed after history, physical examination, and ECG, it is reasonable to obtain a BNP or proBNP and hs-cTnT to aid recognition of cardiac syncope and for risk stratification.[66-68]
- Patients who do not require active treatment are generally appropriate for observation with monitoring, even those with high-risk features.
- Many clinical risk-stratification tools have commonalities **(see Table 8, page 11)**, but at this time, none of them perform better than clinical judgment in predicting adverse events.
- Approximately one-third of patients who present to the ED with syncope are admitted to the hospital.
- Patients with risk factors by past medical history but with a benign etiology of the syncopal event identified in the ED (such as dehydration) may be discharged safely.[114]
- Approximately 9% of patients may have recurrence of a syncopal episode within 6 months;[116] consult local laws regarding release for driving or return-to-work restrictions.

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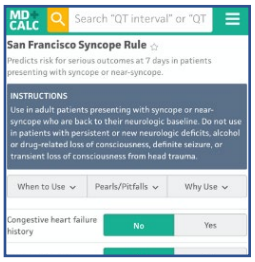
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INSTRUCTIONS
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When to Use
Pearls/Pitfalls
Why Use
Congestive heart failure history
No
Yes

San Francisco Syncope Rule

The San Francisco Syncope Rule predicts risk for serious outcomes at 7 days in patients presenting with syncope or near syncope.

Calculator Review Authors

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About the Score

Syncope may be relatively benign, or it may be a manifestation of serious underlying pathology. The gestalt of the emergency clinician is highly sensitive but poorly specific for predicting high-risk patients. The creators of the San Francisco Syncope Rule designed it to improve specificity while maintaining high levels of sensitivity. This tool can be utilized to predict low-risk syncope patients who are unlikely to benefit from aggressive workup and hospitalization, thus reducing unnecessary testing, healthcare costs, and potential harms associated with hospitalization.

The most common serious outcome in the studies of the San Francisco Syncope Rule was cardiac dysrhythmia; most of those were bradydysrhythmias or sick sinus syndrome, and the remainder were supraventricular or ventricular dysrhythmias. Other common serious outcomes in patients misclassified as low-risk were stroke and intracranial hemorrhage. Validation studies were not able to replicate the high sensitivity found in the original studies published by the creators of the rule.

A commonly used mnemonic to recall the 5 variables of the San Francisco Syncope Rule is "CHESS":

- **C**ongestive heart failure history
- **H**ematocrit <30%
- **E**lectrocardiogram (ECG) abnormal
- **S**hortness of breath
- **S**ystolic blood pressure <90 mm Hg

Evidence Appraisal

Since its inception, the San Francisco Syncope Rule has undergone considerable scrutiny and has been evaluated in validation trials and meta-analyses (key studies include Sun 2007 and Birnbaum 2008). The

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initial derivation study (Quinn 2004) was a single-center, prospective cohort study of 684 patients with a chief complaint of syncope or near-syncope. Fifty predictor variables selected from literature review and expert consensus were incorporated into a survey completed by the physician evaluating the patient after a syncopal episode. The patients were followed through hospital admission and/or contacted at 7 days after the initial presentation to assess for serious outcomes. The predictor variables associated with serious outcomes were identified on univariate analysis. Recursive partitioning was used to find the best combination of predictor variables that would maximize specificity while preserving high sensitivity. Once the combination of predictors was identified, the rule was applied to the derivation set of patients, resulting in sensitivity of 96% and specificity of 62%.

The rule was validated by Quinn et al 2 years later with another single-center, prospective cohort study of 760 patients. Physicians treated patients with syncope or near-syncope in their usual manner and were then asked to fill out a questionnaire about the patients, which included the 5 San Francisco Syncope Rule syncope predictors. After following the patients for 30 days, the San Francisco Syncope Rule was able to predict serious outcomes in all but 1 patient (98% sensitivity). The rule had higher specificity than physician judgment (56% specificity), with an estimated decrease in overall admission rate by 7%, if applied. The single missed patient was suspected to have had transient ischemic attack related to extensive carotid and vertebral artery disease requiring a stent.

Several external validation studies and meta-analyses have also examined the San Francisco Syncope Rule since the derivation study. Sun et al externally validated the rule with a single-center, prospective cohort study with a sample size of 477 patients. Using definitions, exclusion criteria, and methods similar to the internal validation study, the authors found a sensitivity of 89% and a specificity of 42%. Of the 6 patients misclassified as low-risk, 3 had dysrhythmias, 1 had a cerebrovascular accident, 1 had an intracerebral hemorrhage, and 1 had hypertrophic obstructive cardiomyopathy. This study also compared physician judgment to the clinical decision rule. Physician judgment had a sensitivity of 100% and a specificity of 30% for predicting serious outcomes at 7 days.

Another single-center, prospective, observational cohort study by Birnbaum et al performed at a large urban academic center found that the San Francisco Syncope Rule did not perform as well as previously reported. The study found the rule had a sensitivity of 74% and specificity of 54% for predicting serious outcomes at 7 days. The rule would have decreased the overall admission rate by 41%, but it misclassified 16 of the 61 patients who had serious outcomes as low-risk. Physician judgment in this study was 100% sensitive. In a written response to the study published in the *Annals of Emergency Medicine*, the derivation study authors criticized the validation study for incorrect application of ECG criteria used to define “high risk” in the study. The original study authors argued that the subjective interpretation of the ECG is enhanced when performed by a treating physician who is familiar with the clinical context. They posited that the sensitivity of the rule in the New York study was reduced because ECGs were evaluated retrospectively by experts rather than by the physician seeing the patient.

Meta-analyses published in the *Annals of Emergency Medicine* and the *Canadian Medical Association Journal* found sensitivity and specificity similar to the external validation study by Sun et al. The *Annals of Emergency Medicine* meta-analysis found 86% sensitivity and 49% specificity, while the *Canadian Medical Association Journal* meta-analysis found 87% sensitivity and 52% specificity. The most commonly missed serious outcome in all of the studies assessing the predictive value of the San Francisco Syncope Rule was cardiac dysrhythmia.

Instructions

Use this score in adult patients presenting with syncope or near-syncope who are back to their neurologic baseline. Do not use it in patients with persistent or new neurologic deficits, alcohol- or drug-related loss of consciousness, definite seizure, or transient loss of consciousness from head trauma.

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Use the Calculator Now

Access the San Francisco Syncope Score on MDCalc

Calculator Creators

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Read more about Dr. Stiell and Dr. Quinn

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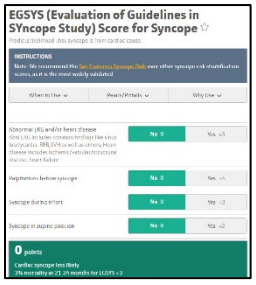

EGSYS (Evaluation of Guidelines in SYncope Study) Score for Syncope

The EGSYS score predicts the likelihood that syncope is from a cardiac cause.

Calculator Review Author

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About the Score

The Evaluation of Guidelines in SYncope Study (EGSYS) score is for use in patients presenting to the emergency department with syncope. The EGSYS authors suggest the score be used to aid emergency clinicians in screening, not as a substitute for clinical judgment in syncope management.

Cardiac syncope carries high mortality if untreated. A score that identifies these patients can help prevent adverse outcomes from underlying cardiac pathology. Patients with suspected cardiogenic syncope should be referred to a cardiologist.

MDCalc recommends the San Francisco Syncope Rule over other syncope risk stratification scores, as it is the most widely validated.

Evidence Appraisal

The EGSYS tool may have limited generalizability. In the original study, a cardiologist specializing in syncope was available to review the electrocardiogram in the emergency setting. The score includes specific criteria for electrocardiogram abnormalities. It is less sensitive (95%) than the San Francisco Syncope Rule (96%-98%).

Use the Calculator Now

Access the San Francisco Syncope Score on MDCalc

Calculator Creator

Attilio Del Rosso, MD
Read more about Dr. Rosso

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Original/Primary Reference

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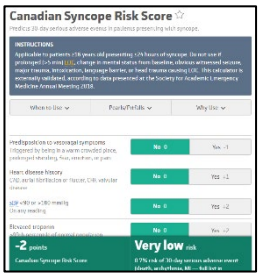

Canadian Syncope Risk Score

The Canadian Syncope Risk Score predicts 30-day serious adverse events in patients presenting with syncope.

Calculator Review Authors

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About the Score

Syncope is a common presenting complaint in emergency departments (EDs); it is often benign but sometimes has life-threatening underlying causes. The Canadian Syncope Risk Score (CSRS) identifies patients with syncope who are at higher risk for adverse outcomes within 30 days. Adverse events are defined as any of the following: death, arrhythmia, myocardial infarction, serious structural heart disease, aortic dissection, pulmonary embolism, severe pulmonary hypertension, severe hemorrhage, subarachnoid hemorrhage, or other serious conditions that caused syncope and/or required interventional treatment of syncope. The tool can potentially help avoid unnecessary investigation and/or admission. The CSRS was successfully validated in 2020.

The tool should be used in patients aged ≥16 years who present within 24 hours of syncope. Do not use the CSRS in patients who meet any of the following criteria: prolonged (>5 min) loss of consciousness (LOC), change in mental status from baseline, obvious witnessed seizure, major trauma requiring hospital admission, intoxication with alcohol or illicit drugs, language barrier, or head trauma causing LOC. Patients at medium or high risk (CSRS score of ≥1 point) should undergo further investigation for cardiac and noncardiac causes of syncope.

Evidence Appraisal

The CSRS was derived by Thiruganasambandamoorthy et al in a prospective cohort study based on 6 large Canadian EDs. The study enrolled 4030 patients with syncope, with a mean age of 53.6 years; 55.5% of the patients were women. Forty-three predictors of serious adverse events were studied, with 9 included in the final model. A “serious adverse event” was defined as death, arrhythmia, myocardial infarction, serious structural heart disease, aortic dissection, pulmonary embolism, severe pulmonary hypertension, severe hemorrhage, subarachnoid hemorrhage, or any other serious condition causing syncope and procedural interventions for the treatment of syncope. The study found that 3.6% of patients sustained a serious adverse event within 30 days of disposition from the ED.

On internal validation, scores ≥-2 were found to have 99% sensitivity and 26% specificity. Scores ≥-1 had 98% sensitivity and 46% specificity. A secondary validation in a group of Canadian EDs found that patients in the low-risk and very low-risk categories appeared to have even lower actual risk percentages (0.7% and 0.3%, respectively).

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Use the Calculator Now

Access the Canadian Syncope Risk Score on MDCalc

Calculator Creator

Venkatesh Thiruganasambandamoorthy, MBBS, CCFP-EM, MSc
Read more about Dr. Thiruganasambandamoorthy

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Original/Primary Reference

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Validation Reference

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